JACC Vol 4, No. 1
July 1984:39–44

Clinical Features and Prognosis of Patients With Out of Hospital Cardiac Arrest and a Normal Electrophysiologic Study

FRED MORADY, MD, FACC,* LORENZO DiCARLO, MD, STUART WINSTON, DO, JESSE C. DAVIS, MD, MELVIN M. SCHEINMAN, MD, FACC

San Francisco, California

Nineteen patients survived a cardiac arrest not associated with an acute myocardial infarction, and had a normal electrophysiologic study with no inducible ventricular tachycardia despite programmed stimulation with one to three extrastimuli at two or more ventricular sites. Among 14 patients who had obstructive coronary artery disease, cardiac arrest occurred during exertion or an episode of angina pectoris in 11; 24 hour ambulatory electrocardiographic recordings demonstrated infrequent or no premature ventricular complexes in 10 and an ischemic response occurred during stage I or II (Bruce protocol) in 6 of 9 patients who underwent exercise testing. Treatment of these patients consisted of myocardial revascularization (eight patients) or antianginal medications (six patients). Only three patients were also treated with an antiarrhythmic drug. Over a follow-up period of 26 ± 15 months (mean ± standard deviation), only one patient died suddenly.

Two patients who had coronary artery spasm were treated with coronary vasodilator medications and had no recurrence of cardiac arrest over 7 and 36 months of follow-up, respectively. Three patients who had cardiomyopathy or no identifiable structural heart disease were treated with nadolol or amiodarone and had no recurrence of cardiac arrest over 3 to 27 months of follow-up.

Among patients who survive a cardiac arrest and have a normal electrophysiologic study, those with obstructive coronary artery disease or coronary artery spasm generally have an excellent prognosis with treatment directed primarily at the underlying heart disease. The clinical features of these patients suggest that cardiac arrest was related to ischemia rather than a primary arrhythmia. The natural history of patients with a cardiomyopathy or no identifiable structural heart disease who survive a cardiac arrest and have a normal electrophysiologic study remains to be determined.

Ventricular tachycardia can be induced by programmed ventricular stimulation in the majority of patients who survive an out of hospital episode of cardiac arrest unassociated with an acute myocardial infarction (1–5). Long-term antiarrhythmic drug treatment of these patients guided by the results of electropharmacologic testing has been associated with a low incidence of sudden death (1,3–5).

In contrast, of the patients who survive a cardiac arrest with documented ventricular tachycardia or ventricular fibrillation at the time of resuscitation, 12 to 37% do not have inducible ventricular tachycardia or ventricular fibrillation during electrophysiologic testing (1–5). The prognosis in these patients is unclear. Although some investigators (1,3,6) have reported that such patients have an excellent prognosis without specific antiarrhythmic drug therapy but with treatment of the underlying heart disease, others (4,7) have reported that 32 to 50% of these patients die suddenly, either with or without antiarrhythmic drug treatment.

The purpose of this study is to describe the clinical features and prognosis of patients who survived an isolated episode of cardiac arrest and who did not have inducible ventricular tachycardia or other abnormalities during electrophysiologic testing.

Methods

Patients. Between June 1979 and August 1983, 96 patients underwent an electrophysiologic study at Moffitt Hospital because of one or more episodes of out of hospital cardiac arrest. Nineteen of these 96 patients who had no demonstrable abnormalities during electrophysiologic testing are the subjects of this report.

All 19 patients had a history of a cardiac arrest with sudden collapse and complete loss of consciousness that

From the Department of Medicine and Cardiovascular Research Institute, University of California, San Francisco, California. Manuscript received November 15, 1983; revised manuscript received January 30, 1984, accepted February 2, 1984.

*Present address and address for reprints: Fred Morady, MD, Room W 11511, University Hospital, University of Michigan, Ann Arbor, Michigan 48109.

0735-1097/84/$3.00

required cardiopulmonary resuscitation. In all patients, the cardiac rhythm at the time of direct current countershock was ventricular fibrillation or ventricular flutter. Although 16 of the 19 patients had mild elevation of the serum creatine kinase MB fraction, none had electrocardiographic evidence of an acute myocardial infarction (new Q waves or persistent ST segment/T wave abnormalities). In addition, no patient had myocardial uptake on a technetium pyrophosphate myocardial scintigram performed 1 to 3 days after the cardiac arrest.

The following exclusionary criteria were applied in selecting patients for this study: 1) a high suspicion of drug-induced ventricular tachycardia, that is, cardiac arrest occurring in a patient treated with a type I antiarrhythmic drug, with recurrent episodes of ventricular tachycardia of the "torsade de pointes" type (8) and a prolonged QT interval; 2) cardiac arrest associated with a metabolic abnormality; and 3) cardiac arrest occurring as a terminal event in patients with cardiogenic shock. Because our intent was to evaluate patients with an isolated episode of cardiac arrest, we also excluded patients with documented ventricular tachycardia in whom ventricular fibrillation was caused by a recurrent episode of ventricular tachycardia.

Noninvasive studies. The patients were evaluated 1 to 6 weeks after their cardiac arrest. An exercise treadmill test (Bruce protocol) was performed in 14 patients (with thallium-201 scintigraphy in 12); 5 patients did not undergo treadmill testing because of unstable angina or the inability to perform upright exercise. An ischemic response to exercise testing was defined as 1 mm or greater horizontal ST segment depression at 80 ms after the J point or a perfusion defect with redistribution on delayed scintigrams obtained 2 to 3 hours after the exercise study.

Continuous electrocardiographic monitoring in the absence of antiarrhythmic drug therapy was performed for at least 24 hours in all patients. The following classification system, described by Lown and Wolf (9), was used to grade premature ventricular complexes: grade 0 = no premature ventricular complexes; grade 1 = less than 30 premature ventricular complexes per hour (1A = less than 1 per minute; 1B = more than 1 per minute); grade 2 = more than 30 premature ventricular complexes per hour; grade 3 = multiform premature ventricular complexes; grade 4 = repetitive premature ventricular complexes (4A = ventricular couplets; 4B = three or more consecutive premature ventricular complexes); and grade 5 = early premature ventricular complexes (R on T). A two-dimensional echocardiogram was performed in all patients.

Cardiac catheterization. All patients, with the exception of one who refused, underwent cardiac catheterization including left ventricular and coronary angiography. An ergonovine provocation test (10) was performed if obstructive coronary artery lesions were not seen on the coronary angiograms.

Electrophysiologic testing. Five patients who had severe coronary artery disease underwent uneventful coronary artery bypass graft surgery 7 to 10 days before electrophysiologic testing. One patient underwent electrophysiologic testing before and after bypass surgery (with both studies being negative). Electrophysiologic testing was performed with the patient in the fasting unsedated state after informed written consent was obtained, at least five half-lives after discontinuation of antiarrhythmic drugs. Pacing stimuli were twice diastolic threshold in the first five patients studied and 5 mA in subsequent patients. The stimuli were 2 ms in duration and were delivered by a programmable stimulator (Bloom Associates).

The following stimulation protocol was used. Overdrive pacing was performed for 5 to 10 seconds at cycle lengths of 500 to 250 ms. Programmed ventricular stimulation was performed with one, two and three ventricular extrastimuli at drive trains of 500 and 400 ms. The stimulation protocol was performed initially at the apex of the right ventricle. In all patients, the stimulation protocol was then repeated at one or more additional ventricular sites (right ventricular outflow tract in five patients, left ventricular apex in eight patients and both the right ventricular outflow tract and left ventricular apex in six patients). In the five patients who did not have obstructive coronary artery disease, the stimulation protocol was repeated at one or two ventricular sites during a continuous infusion of isoproterenol, titrated to maintain a heart rate of 120 beats/min.

Sustained ventricular tachycardia was defined as ventricular tachycardia lasting at least 30 seconds or requiring electrical cardioversion. Nonsustained ventricular tachycardia was defined as ventricular tachycardia six beats to 30 seconds in duration. No patient in this study had inducible sustained or nonsustained ventricular tachycardia. In 17 of the patients, one to five repetitive ventricular complexes were induced.

The electrophysiologic testing protocol also included measurement of the sinus node recovery time, assessment of atrioventricular conduction and attempts to induce supraventricular tachycardia. Results of testing were noncontributory in all patients.

Follow-up. The patients were followed up by either one of us or the referring physicians. A 24 hour ambulatory electrocardiographic recording was obtained every 3 to 6 months. Sudden death was defined as unexpected death from natural causes occurring within 1 hour of the patient's collapse.

Results

Patients With Obstructive Coronary Artery Disease

Clinical features. There were 12 men and 2 women with a mean age of 62 ± 12 years (mean ± standard deviation) (Table 1). Twelve of the 14 patients had a known history

Table 1. Clinical Features of 19 Patients Who Survived Cardiac Arrest and Had a Normal Electrophysiologic Study

Case	Age (yr) & Sex	Heart Disease	EF	PVC Grade†	Treadmill Test*		
					Ischemic Response	Stage Reached	PVC Grade†
1	54M	CAD (3v)	0.50	0	+	III	VT
2	72M	CAD (2v)	0.55	1A	ND		
3	70M	CAD (3v)	0.45	1A	+	I	1A
4	80F	CAD (LM)	0.73	1A	ND		
5	71M	CAD‡	0.56	0	+	II	0
6	62M	CAD (3v)	0.40	2	ND		
7	63M	CAD (3v)	0.25	1B	ND		
8	69M	CAD (LM)	0.20	3	+	I	3
9	45M	CAD (2v)	0.69	0	+	III	0
10	61M	CAD (3v)	0.64	3	+	II	4A
11	55M	CAD (1v)	0.44	1A	+	II	0
12	73M	CAD (3v)	0.77	1A	+	II	2
13	40F	CAD (2v)	0.60	0	+	III	0
14	48M	CAD (3v)	0.20	2	ND		
15	50M	Spasm	0.80	0	+	IV	0
16	62M	Spasm	0.56	4A	−	III	0
17	58M	HCM	0.60	0	−	IV	0
18	61F	CCM	0.32	2	−	II	2
19	33M	None	0.72	0	−	V	4A

*Bruce protocol; †see text for premature ventricular complex classification system; ‡coronary angiography not performed. CAD = coronary artery disease; CCM = congestive cardiomyopathy; EF = left ventricular ejection fraction; HCM = hypertrophic cardiomyopathy; LM = left main coronary artery; ND = not determined; PVC = premature ventricular complex; VT = sustained ventricular tachycardia; 1v = single vessel; 2v = double vessel; 3v = triple vessel; + = present; − = absent.

of angina pectoris or myocardial infarction, or both. None had undergone coronary artery bypass graft surgery before cardiac arrest. Only two patients had overt congestive heart failure. At the time of cardiac arrest, seven patients were being treated with propranolol (80 to 160 mg/day) and nitrates for angina pectoris, and one was being treated with procainamide (1.5 g/day) because of frequent premature ventricular complexes.

Cardiac arrest occurred during strenous exertion (bicycling, running or carrying a heavy package) in seven patients and during mild exertion (walking) in one. These patients all had retrograde amnesia and did not recall whether they experienced chest pain before the cardiac arrest. In an additional three patients, the cardiac arrest was preceded by chest pain typical of angina pectoris, occurring at rest or during minimal exertion.

Catheterization findings (Table 1). Among the 13 patients who underwent coronary angiography, 2 had a significant (> 70%) stenosis of the left main coronary artery, 7 had triple vessel disease, 3 had double vessel disease and only 1 had single vessel disease. The mean left ventricular ejection fraction (determined by contrast left ventriculography in 13 patients and radionuclide angiography in 1) was 0.50 ± 0.19. Thirteen patients had one or more ventricular wall motion abnormalities; nine of these patients had a discrete ventricular aneurysm.

Electrocardiographic findings (Table 1). The 24 hour ambulatory electrocardiographic recording demonstrated grade 0 or 1 premature ventricular complexes in 10 patients and grade 2 or 3 premature ventricular complexes in 4 patients.

An ischemic response occurred in all nine patients who underwent an exercise treadmill test. During exercise, one patient developed sustained ventricular tachycardia that required electrical cardioversion and only two other patients had an increase in grade of premature ventricular complexes above the baseline value.

Follow-up (Table 2). The mean duration of follow-up was 26 ± 15 months. Seven patients underwent coronary artery bypass graft surgery and one patient underwent percutaneous transluminal coronary angioplasty. These patients were not treated with antiarrhythmic drugs; none has had a recurrence of cardiac arrest and all remain alive and well, except for one patient who died of pneumonia. Two patients who had frequent premature ventricular complexes were treated with either procainamide or encainide in addition to antianginal medications. Follow-up Holter monitor recordings demonstrated greater than 85% suppression of premature ventricular complexes in both. The patient who was being treated with encainide died suddenly after a heated argument at 15 months of follow-up. A nitroglycerin tablet was found in his hand; an autopsy was not performed. The remaining four patients were treated with antianginal medications and have remained well without a recurrence of cardiac arrest. Only one of the latter patients was also treated empirically with an antiarrhythmic drug (amiodarone) at the request of his referring physician.

Patients Without Obstructive Coronary Artery Disease

Clinical findings (Table 1). There were four men and one woman who did not have coronary artery disease. Their mean age (± standard deviation) was 53 ± 12 years. Two patients had a positive ergonovine provocation test for focal coronary artery spasm, one patient had a nonobstructive hypertrophic cardiomyopathy, one had congestive cardiomyopathy and one had no demonstrable structural heart disease. The left ventricular ejection fraction was normal in all patients, except one who had congestive cardiomyopathy and an ejection fraction of 0.32.

The cardiac arrest in both patients who had coronary artery spasm was preceded by chest pain that occurred in one patient during running and in the other while at rest. In the other three patients, the cardiac arrest occurred during rest or minimal exertion. One patient was being treated with quinidine at the time of the cardiac arrest because of frequent premature ventricular complexes.

Electrocardiographic findings (Table 1). Three patients had no premature ventricular complexes (grade 0) during a 24 hour ambulatory electrocardiographic recording, while one had frequent grade 2 and one had grade 4 premature ventricular complexes (Table 1).

The patient with coronary artery spasm who had chest pain and cardiac arrest while running had an ischemic response during exercise testing, but no ventricular ectopic activity. The other four patients did not have chest pain or ST segment abnormalities during the exercise treadmill test. The premature ventricular complex grade was greater during exercise than at baseline in only one of the five patients.

Follow-up (Table 2). The mean follow-up period was 16 ± 15 months in these five patients. The two patients who had coronary artery spasm were treated with a calcium channel blocking agent, in addition to propafenone in the patient who had frequent premature ventricular complexes. The other three patients were treated empirically with nadolol or amiodarone. All have remained well without a recurrence of cardiac arrest.

Discussion

Prognosis. Fourteen of the 19 patients in this series who survived cardiac arrest and had a normal electrophysiologic study had coronary artery disease. Only 3 of the 14 were treated with an antiarrhythmic drug; the others either underwent coronary artery bypass graft surgery or were treated with antianginal medications. Only 1 of the 14 patients died suddenly over a mean follow-up period of 26 months. These findings demonstrate that patients with coronary artery disease who survive a cardiac arrest and have a negative electrophysiologic study may have an excellent prognosis with treatment directed only toward the coronary artery disease and do not necessarily require specific antiarrhythmic drug therapy.

These results confirm the preliminary findings of Tommaso et al. (6), who reported that 16 survivors of cardiac arrest who did not have inducible ventricular tachycardia and were treated only with bypass surgery or beta-adrenergic blocking agents had no recurrence of cardiac arrest over 14

Table 2. Treatment and Follow-up of 19 Patients Who Survived Cardiac Arrest and Had a Normal Electrophysiologic Study

Case	Revascularization Procedure	Drug Therapy		Follow-up (mo)
		Antianginal	Antiarrhythmic	
1	CABG	—	—	54
2	CABG	—	—	47
3	CABG	—	—	45
4	CABG	—	—	39
5	—	Nadolol, nitrates	—	31
6	—	Nifedipine, nitrates	Procainamide	27
7	CABG	—	—	26 (died of pneumonia)
8	CABG	—	—	24
9	—	Nadolol, nitrates	—	20
10	—	Propranolol, nitrates	Encainide	15 (sudden death)
11	—	Nitrates	Amiodarone	14
12	—	Propranolol, nitrates	—	14
13	PTCA	—	—	6
14	CABG	—	—	5
15	—	Nifedipine	—	36
16	—	Verapamil	Propafenone	17
17	—	—	Amiodarone	5
18	—	—	Amiodarone	3
19	—	—	Nadolol	27

CABG = coronary artery bypass grafting; PTCA = percutaneous transluminal coronary angioplasty.

months of follow-up. In addition, our results confirm an earlier report from our laboratory (3) in which seven patients who had coronary artery disease and no inducible ventricular tachycardia had no recurrence of cardiac arrest with conventional treatment of the coronary disease. Ruskin et al. (1) also reported that two patients with coronary artery disease who survived cardiac arrest and had no inducible ventricular tachycardia did well after bypass surgery, without antiarrhythmic drug therapy.

In contrast, Roy et al. (7) reported that survivors of cardiac arrest who did not have inducible sustained ventricular tachycardia did not have a good prognosis, with a 32% incidence of sudden death over a mean follow-up period of 20 months. However, in their preliminary report, Roy et al. did not report results in the subgroup of patients with coronary artery disease. In addition, although their patients did not have sustained ventricular tachycardia, many may have had inducible nonsustained ventricular tachycardia. In our study and the studies mentioned earlier (1,6), the patients had neither inducible sustained nor nonsustained ventricular tachycardia. Inducible nonsustained ventricular tachycardia may have different prognostic implications than no inducible ventricular tachycardia.

Role of coronary artery disease and ischemia. Our patients with coronary artery disease showed the following general characteristics: 1) the coronary artery disease was usually severe, with 9 of 13 patients who underwent cardiac catheterization having disease of the left main coronary artery or triple vessel disease; 2) all the patients who underwent treadmill testing had an ischemic response in stage I, II or III (Bruce protocol); and 3) 10 of the 14 patients were engaged in strenuous activity or experienced angina pectoris at rest at the time of the cardiac arrest. Because of retrograde amnesia, it is not known whether the remaining patients had angina at the time of their cardiac arrest. These general characteristics, in association with the finding of a negative electrophysiologic study, suggest that the cardiac arrest in these patients was caused by ventricular tachycardia or ventricular fibrillation, precipitated by transient ischemia. The finding of noninducible ventricular tachycardia after cardiac arrest suggests that the substrate for reentry was not present at the time of electrophysiologic testing. It may be that the appropriate electrophysiologic milieu for ventricular tachycardia or ventricular fibrillation to occur in these patients was dependent on the presence of myocardial ischemia. This possibility is supported by the finding that our patients with coronary artery disease and a negative electrophysiologic study had an excellent prognosis when treatment was directed only toward the prevention of myocardial ischemia.

Benditt et al. (4) reported that of two patients with coronary artery disease who survived a cardiac arrest and had a negative electrophysiologic study, one died suddenly despite undergoing coronary artery bypass surgery and treatment with propranolol. In addition, 1 of our 14 patients died suddenly despite treatment with antianginal medications. Therefore, a negative electrophysiologic study in patients with coronary artery disease does not guarantee a benign prognosis. It may be that the patients who died suddenly in our series and in the series of Benditt et al. (4) had a recurrence of myocardial ischemia, which precipitated a malignant ventricular arrhythmia. Consistent with this possibility is that our patient who died suddenly was found with a nitroglycerin tablet in his hand, suggesting that his death may have been preceded by angina pectoris.

Role of myocardial revascularization. Garan et al. (11) reported that among 17 patients with coronary artery disease who had cardiac arrest or ventricular tachycardia, ventricular tachycardia was inducible in 15 patients before myocardial revascularization but in only 7 patients after revascularization. This finding demonstrates that treatment of myocardial ischemia alone may eliminate the potential for ventricular tachycardia or ventricular fibrillation in some patients with coronary artery disease. Five of our patients underwent electrophysiologic testing only after having undergone myocardial revascularization. The findings of Garan et al. (11) suggest that some of these patients may have had inducible ventricular tachycardia or ventricular fibrillation before myocardial revascularization. However, the one patient in our study who underwent electrophysiologic testing both before and after myocardial revascularization did not have inducible ventricular tachycardia during either study.

Role of exercise. Although all of our patients with coronary artery disease who underwent treadmill testing had an ischemic response, only one developed sustained ventricular tachycardia. It may be that ventricular tachycardia or ventricular fibrillation did not occur during exercise in the remaining patients because the testing was stopped as soon as signs or symptoms of ischemia occurred. In addition, the development of ventricular tachycardia or ventricular fibrillation in patients who experience myocardial ischemia during exercise may depend on the chance occurrence of premature ventricular complexes that provide the appropriate trigger for the ventricular tachycardia or ventricular fibrillation.

Role of coronary spasm. Although 5 of the 19 patients in this study did not have coronary artery disease, myocardial ischemia could be implicated as a causal factor of cardiac arrest in an additional 2 of these 5 patients as a result of artery spasm. These patients have had no recurrence of cardiac arrest (or chest pain) while being treated with calcium channel blocking agents.

Role of cardiomyopathy. The remaining three patients in this series had either a hypertrophic cardiomyopathy, congestive cardiomyopathy or no demonstrable structural heart disease. In these patients, no potential precipitant of cardiac arrest could be identified. Although subendocardial ischemia may theoretically occur in patients with cardio-

myopathy who do not have coronary artery disease, these patients did not demonstrate any signs or symptoms of ischemia during exercise testing. Because no potentially remediable precipitant of cardiac arrest was identifiable and because of the fear of a recurrence of cardiac arrest, these patients were treated empirically with amiodarone or nadolol. The natural history of patients with cardiomyopathy or without identifiable structural heart disease who survive cardiac arrest and have a negative electrophysiologic study remains unknown.

Role of antiarrhythmic drugs. Graboys et al. (12) reported that the abolition of advanced grades of premature ventricular complexes with antiarrhythmic drugs in patients who had ventricular fibrillation or ventricular tachycardia provided a high degree of protection against sudden death. Of note is that only 9 of 19 patients in our study had advanced grades of premature ventricular complexes (grades 2 to 5) during ambulatory electrocardiographic recordings or exercise testing. Therefore, the technique of premature ventricular complex suppression described by Graboys et al. would not have been applicable in a large proportion of patients in this series. The one patient in our study who died suddenly had frequent premature ventricular complexes and couplets, with more than 90% suppression by encainide. Therefore, in the subgroup of patients with coronary artery disease who do not have inducible ventricular tachycardia, suppression of premature ventricular complexes may not protect against sudden death.

The stimulation protocol used in our study was an aggressive one that included the use of triple ventricular extrastimuli and stimulation at multiple ventricular sites. The use of triple extrastimuli and multiple ventricular sites increases the yield of programmed ventricular stimulation. Therefore, our results may not apply to patients who undergo electrophysiologic testing with a less aggressive stimulation protocol.

Conclusion. Electrophysiologic testing plays an important role in the evaluation of patients who have survived cardiac arrest. A normal electrophysiologic study appears to identify those patients with coronary artery disease who generally have an excellent prognosis with treatment directed primarily at the prevention of ischemia. In contrast, as prior studies (1,3,4) have demonstrated, the finding of inducible ventricular tachycardia indicates the need for specific antiarrhythmic therapy in addition to treatment of the underlying heart disease.

In the subgroup of patients who survive cardiac arrest and do not have coronary artery disease, electropharmacologic testing can be performed if ventricular tachycardia is inducible. In those who do not have inducible ventricular tachycardia, specific recommendations cannot be made regarding the need for long-term antiarrhythmic drug therapy. Because of the fear of recurrent cardiac arrest, we have treated these patients empirically with antiarrhythmic drugs; their natural history remains unclear.

References

1. Ruskin JN, DiMarco JP, Garan H. Out of hospital cardiac arrest. Electrophysiologic observations and selection of long-term antiarrhythmic therapy. N Engl J Med 1980;303:607–13.
2. Josephson ME, Horowitz LN, Spielman SR, Greenspan AM. Electrophysiologic and hemodynamic studies in patients resuscitated from cardiac arrest. Am J Cardiol 1980;46:948–55.
3. Morady F, Scheinman MM, Hess DS, Sung RJ, Shen E, Shapiro W. Electrophysiologic testing in the management of survivors of out-of-hospital cardiac arrest. Am J Cardiol 1983;51:85–9.
4. Benditt DG, Benson DW Jr, Klein GJ, Pritzker MR, Kriett JM, Anderson RW. Prevention of recurrent sudden cardiac arrest: role of provocative electropharmacologic testing. J Am Coll Cardiol 1983;2:418–25.
5. Ruskin JN, Garan H, DiMarco JP, Kelly E. Electrophysiologic testing in survivors of prehospital cardiac arrest: therapy and long-term follow-up (abstr). Am J Cardiol 1982;49:958.
6. Tommaso C, Kehoe R, Zheutlin T. Survivors of ischemic mediated sudden death—clinical, angiographic and electrophysiologic features and response to therapy (abstr). Circulation 1982;66(suppl II):II-25.
7. Roy D, Waxman HL, Kienzle MG, Pembrook-Rogers D, Josephson ME. Clinical characteristics and long term follow up in survivors of cardiac arrest: relation to inducibility at electrophysiologic evaluation (abstr). Circulation 1982;66(suppl II):II-25.
8. Krikler DM, Curry PVL. Torsade de pointes, an atypical ventricular tachycardia. Br Heart J 1976;38:117–20.
9. Lown B, Wolf M. Approaches to sudden death from coronary heart disease. Circulation 1971;44:130–42.
10. Schroeder JS, Bolen JL, Quint RA, et al. Provocation of coronary spasm with ergonovine maleate. New test with results in 57 patients undergoing coronary arteriography. Am J Cardiol 1977;40:487–91.
11. Garan H, Ruskin JN, DiMarco JP, et al. Electrophysiologic studies before and after myocardial revascularization in patients with life-threatening ventricular arrhythmias. Am J Cardiol 1983;51:519–24.
12. Graboys TB, Lown B, Podrid PJ, DeSilva R. Long-term survival of patients with malignant ventricular arrhythmia treated with antiarrhythmic drugs. Am J Cardiol 1982;50:437–43.